

REMARKS:

Applicants request reconsideration and reexamination of the subject application, pursuant to and consistent with 37 C.F.R. §1.116, and in light of the remarks which follow.

At the outset, Applicants apologize for the delay in submitting the comparative data contained in the attached §132 Declaration by Darrell Anderson, Ph.D. However, this delay was unavoidable as there was a several month back order with respect to obtaining the de Boer cell line from the American Type Culture Collection. Any inconvenience this causes the Examiner is regretted.

Turning now to the Office Action, the only rejection that remains outstanding is a §102(e) rejection based on de Boer, U.S. Patent 5,747,034. The Office Action essentially holds that absent further objective evidence it cannot be determined whether the anti-B7.1 antibody disclosed in the de Boer patent inherently possesses the same binding specificity as the claimed anti-B7.1 antibody.

As previously argued, and reflected by the claims, the inventive anti-B7 antibodies specifically bind to human B7.1 antigen (CD80) and inhibit the binding of B7.1 antigen to CD28, but do not inhibit the binding of B7.1 antigen to CTLA-4.

This is a non-obvious distinction as to Applicants' knowledge the present inventors have obtained the only antibody specific to human B7.1 which does not inhibit the

binding of B7.1 antigen to CTLA-4. This difference is supported by binding data and comparative binding data contained the as-filed disclosure.

Moreover, this non-obvious difference is further substantiated by the additional comparative data contained in the unexecuted §132 Declaration of Darrell Anderson, provided herewith. Contained in this Declaration are the results of ELISAs that compare the binding of several anti-B7.1 antibodies, i.e., B7-24 (de Boer antibody), p16C10 (antibody according to invention), and L307.4 (another known anti-B7.1 antibody). These results clearly show that only p16C10 did not inhibit the binding of B7.1 antigen to CTLA-4.

In these experiments, an ELISA plate was coated with CTLA4Ig, which was then serially diluted with B7-24, p16C10, or L307.4, and mixed with B7Ig-Bio. The results contained in Figure 1 are dramatic in the fact that only p16C10 (the inventive antibody) had no effect on B7Ig binding to CTLA4Ig.

Also, Figure 2 of the Declaration compares the effect of B7-24 and p16C10 on B7 binding. It can be seen from Figure 2, on a B7 Ig coated plate, that serial dilution of B7-24 and p16C10, mixed with p16C10 - Bio, resulted in B7-24 having no effect on the binding of p16C10 to B7.1.

Therefore, it is apparent that the deBoer antibody binds to a completely distinct epitope than p16C10, an antibody according to the invention which, unlike B7-24, does not inhibit the binding of B7.1 to CTLA-4.

Therefore, based on the foregoing, withdrawal of the §102/103 rejection based on de Boer is respectfully requested.

Respectfully submitted,

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